Editorial

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Exercise heart rate/ST segment relation
Perfect predictor of coronary disease?

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Since the introduction of coronary angiography there has been keen interest in trying to find a non-invasive test that will accurately predict the coronary anatomy found at angiography. The results of American and European trials suggesting that in select groups prognosis may be improved by surgery have if anything increased the urgency of finding this cardiological "pot of gold".[1 2]

The most favoured approach has been to develop the exercise test originally described by Master and Oppenheimer.[3] Refinements using a bicycle or motorised treadmill, increased number of leads, and computerisation appeared to bring us closer to our ultimate goal. Not surprisingly, however, there were always discrepancies, since exercise-induced ST segment depression provided a marker of myocardial ischaemia only indirectly related to coronary anatomy.

Thus, it is with extreme interest that we read the article by Elamin and his co-workers in this issue of the *British Heart Journal*.[4] Certainly their approach to the problem differs from any other previously published. On the assumption that the heart rate is the most accurate simple assessment of myocardial oxygen demand and that ST segment depression represents myocardial ischaemia in a semiquantitative manner, the rate of development of myocardial ischaemia with respect to increases in myocardial oxygen demand has been tested as a guide to the severity of coronary anatomy. Other things being equal it may be expected that, broadly, those patients with the most severe coronary disease will develop the most severe myocardial ischaemia (ST segment depression) as myocardial oxygen demand (heart rate) is gradually increased. The most surprising thing about the results of this study is that the relation is not broad at all, but very exact. It was possible to predict exactly not only the presence or absence of coronary disease but also the severity in terms of one, two, and three vessel involvement. The differences found with one, two, and three vessel disease were such that there was no overlap at all in the ST/heart rate relation. This is all the more startling when one considers that the tools, that is heart rate as a measure of myocardial oxygen demand, ST segment depression as an assessment of severity of myocardial ischaemia, and even coronary angiography with its inter- and intraobserver variability to define coronary anatomy, are no more than rough guides. Furthermore, to explain these findings requires that we rethink many of our traditional concepts of coronary disease.

If these results are confirmed in different groups and types of patients then coronary collaterals must play no significant role in providing blood supply to areas of myocardium supplied by diseased vessels. Though there is still conflicting evidence presented from animal experiments and clinical studies on the importance of collaterals,[5 6] it is hard to believe that large collaterals, which at angiography fill retrogradely large portions of the coronary tree, play no significant role in providing regional myocardial perfusion. The problem may be one of patient selection.

Similarly, to explain the predictable step increase of heart rate/ST segment relation on the basis simply of single, two, and three vessel disease calls into question many fundamental concepts. Each vessel be it right coronary, left anterior descending, or circumflex, and each patient must function in a completely homogeneous manner. Factors such as vessel dominance, the number, site, severity (narrowing greater than 75%), and length of coronary strictures in each coronary vessel must all play no significant role in this relation between myocardial ischaemia and myocardial oxygen demand. Such biological homogeneity must be almost unique. Again, patient selection may be at the heart of the problem.

This study showing a perfect correlation between measurements taken during exercise and coronary angiograms recorded at rest will also have to be reconciled with many *variable* regulators of coronary flow that have been postulated. During exercise and particularly during the induction of ischaemia it has been suggested that there are many factors that may not be prominent at rest. For example, coronary spasm,[7] passive coronary collapse resulting from fall in perfusion,[8] or physiological alterations in coronary tone[9] may all play a greater or lesser role in determining

coronary flow and thus myocardial ischaemia.

Perhaps this test heralds a new age in cardiology, but before rethinking established physiological concepts of coronary disease, in the words of Lord Asquith "we had better wait and see".

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